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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

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Volume IX TORONTO, FEBRUARY, 1932 Number 4

J. G. FitzGerald, M.B., M.D. Tor., LL.D.,

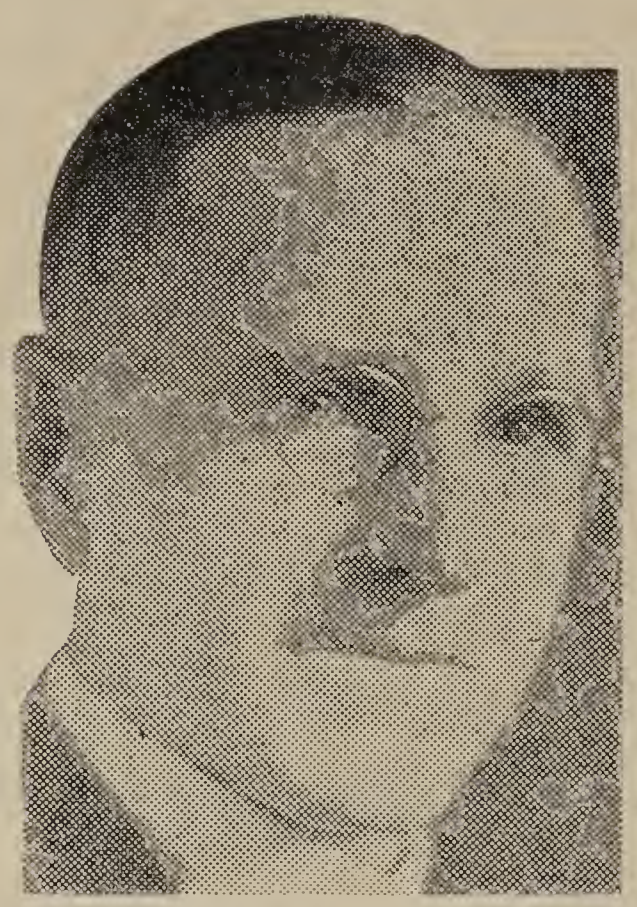
DEAN OF THE FACULTY OF MEDICINE, UNIVERSITY OF TORONTO.

John Gerald FitzGerald was born in Drayton, Ontario, in 1882, received his preliminary education in Harriston, and graduated from the Faculty of Medicine, University of Toronto, in 1903, the youngest member of his class.

After several years spent in psychiatry and neuropathology in Toronto, at the Harvard Medical School, in Baltimore and in other centres, Dr. FitzGerald's interest turned to Bacteriology. In 1909 he was appointed Lecturer in Bacteriology in the University of Toronto, and he spent the next two years doing research work at the Pasteur Institute in Paris and Brussels, and at the University of Frieburg.

From 1911 to 1913 he was Associate Professor of Bacteriology in the University of California, and in 1913 was appointed Associate Professor of Hygiene in the University of Toronto, and in 1914 Director of the Antitoxin Laboratory in the Department of Hygiene.

During the Great War, Dr. FitzGerald served as a Captain in the C.A.M.C. and later as a Major in the R.A.M.C. of the British Expeditionary Force,



DEAN J. G. FITZGERALD, M.B., M.D., TOR., LL.D.

being officer in charge of a Mobile Laboratory in France and Assistant Advisor in Pathology, Fifth British Army.

In 1919 he was made Professor of Hygiene and Preventive Medicine in the University of Toronto, and director of the Connaught Laboratories, where antitoxins, sera, vaccines, insulin and other products are made and investigated. In 1922-1923 he occupied the Chair of Bacteriology and Experimental Pathology in the University of California.

In 1923 Professor FitzGerald was made a member of the International Board of Health of the Rockefeller Foundation, which has recently become a division of the Rockefeller Foundation, and of which division he is a Scientific Director. Chiefly through his efforts, the Foundation made a grant in 1925 for the establishment of the School of Hygiene, and a further grant in 1930 for its extension. Professor FitzGerald was made Director of the School on its foundation, and continues to direct investigations being carried on there in the fields of Public Health, Hygiene and Preventive Medicine. On these subjects he has written widely, and is a recognized authority.

His appointment in 1930 to the Health Committee of the League of Nations is a mark of the world-wide reputation which he has attained. The following list testifies to the extent of his activities:—

Fellow of the Royal Society of Canada and Member of Council.

Fellow of the American Public Health Association.

F.R.C.P. and S. (Canada) and Member of Council.

Member of the Dominion Council of Health.

Trustee of the Banting Research Foundation.

Member of the Executives of the Canadian Medical Association, Canadian Public Health Association and Canadian Tuberculosis Association.

Member of the Advisory and Research Committee of the Canadian National Committee of Mental Hygiene.

Honorary Advisor in Public Health Administration, Department of Health, Ontario.

Member Advisory Council of the Ontario Research Foundation.

Member of Associate Committee on Tuberculosis Research of the National Research Council of Canada.

Fellow of Academy of Medicine, Toronto.

On January 1st, 1932, Professor FitzGerald became Dean of the Faculty of Medicine, to succeed Dr. Primrose, who had occupied the position since 1920. Although it was with deep regret that we learned of the retirement of Dr. Primrose, we are very fortunate in having as his worthy successor a man of such wide interests and attainments. In the name of the student body, we take this opportunity to welcome Dean FitzGerald, and to offer sincere co-operation during the following years.

Editorial.

WELCOME TO THE ASSISTANT DEAN.

Dr. Ryerson's success as Secretary to the Faculty of Medicine, and his proved sympathy with the undergraduate in that office, assure us that as Assistant Dean of Medicine he will be as good a friend of the student as he has always been. We welcome the news of his appointment to the higher honour, feeling that we have more than made up for the loss of a respected and loved Secretary, by the acquisition of Dr. Ryerson as Assistant Dean.

IN THIS ISSUE.

The Editors direct the readers' attention to the articles on Acute Appendicitis and Nephritis in this number. It is intended to publish three articles on each of these subjects, the present number containing the pathology, while succeeding papers on the symptomatology, diagnosis, and treatment, will appear in the March and April issues. We feel that such large subjects cannot be treated successfully in a single article, and therefore have arranged that this method of presentation be adopted. The advantages of such a method are recommended for the consideration of those who contemplate writing on such subjects in the future. It is the aim that each of these articles shall be self-contained as far as possible.

TUBERCULOSIS IN MEDICAL STUDENTS.

Dean Pepper, of the Medical School of the University of Pennsylvania, has recently made a report on the incidence of tuberculosis among medical students (in the Journal of the Association of American Medical Colleges). Though his investigations are not yet complete, enough has been done to vitally interest every medical undergraduate. The results are the more startling inasmuch as similar investigations were carried out among arts men, dental students and law seniors.

Among the non-medical students, an average of

12% showed calcified nodules in the lungs or lymph nodes.
5% showed latent apical tuberculosis.
0.4% showed manifest pulmonary tuberculosis.
5.7% showed latent apical lesions and also manifest tuberculosis.

Of the medical students, on the other hand:

16% showed calcified nodules.
10% latent apical lesions.
1.5% manifest pulmonary tuberculosis.
11.5% apical lesions plus manifest pulmonary tuberculosis.

These figures are much higher than one would believe from casual observation, but experienced clinical and X-ray findings are behind them. The writer notes also that the incidence of the disease leaps upward as the student enters the last two years of his course. These students handle material from autopsies made on tuberculosis patients and have bedside clinics on cases of active pulmonary tuberculosis.

Whether these figures are true at the University of Toronto or not, we cannot say,—undoubtedly we have more cases of tuberculosis amongst ourselves than we know. The only solutions presented in the discussion on Dr. Pepper's paper involved the physical examination of all students annually and health insurance paid for by the Medical School.

Perhaps the medico learns early the notorious habits of the practitioner, who, it is said, watches with anxious care the health of everyone but himself. It is significant that the disease which has such a high and debilitating incidence among medical students is one which is bred by exposure to infection, inadequate rest, and irregular, unbalanced meals.

THIS STATE MEDICINE PROBLEM.

The article on this subject in this month's issue will not be the first nor the last that Journal readers will see. Arguments pro and con are to be seen in the publications of every medical society. In the hospital we see only the richest and the poorest receiving the best medical and surgical attendance, while the middle class, the "poor but proud," and the rest of us, muddle along without calling a doctor till it's too late, and spend the price of a couple of hard-earned vacations to pay for one illness. The young medical man has paid, or has had paid for him, the price of at least six years' intensive teaching and study; has worked hard for no pay (even a labourer's) at an internship for one or more years; then, at the peak of his knowledge, can earn but a precarious living, till gray hairs and angina pectoris have made him a more trustworthy object to consult. When patients do come, he cannot afford to take holidays or post-graduate study, 30% of his bills are unpaid, 30% of his patients he expects to treat for nothing. The cost of proper medical care is going up, and the doctor is getting less for more knowledge, more work, more study.

Whether these problems are to be settled by State medicine, or whether they will be settled at all, we know not. Nevertheless, it behooves the medical profession to be ready to take the lead when the issue becomes a political one, as it did in England. Mr. Cathie's article shows us what has happened in England, despite the medical profession's bitter, but disunited, opposition.

A PROBLEM IN ANATOMY.

At the College of Medicine and Dentistry of the West China Union University, situated at Chengtu, Szechwan, almost two thousand miles inland from Shanghai, the Department of Anatomy had a very real problem. Religious beliefs, ancestor worship, superstition, etc., made it very difficult to secure a sufficient supply of bodies for dissection. However, the missionary scientist is accustomed to solving unusual problems. The head of the department got in touch with the leading military general of the district and suggested that the robbers, traitors, bandits, looters, etc., who were executed from time to time might prove highly useful to society, if not during life, at least after death, if their bodies were available for scientific training of the keen young Chinese medical and dental students. The general was quite willing, and so an adequate supply of cadavers was made available.

However, a glance at a typical Chinese executioner's sword is convincing evidence that the exacting demands of science may be frustrated by man's crude methods. In other words dissection of the neck, etc., presented real difficulties, following the executioner's enthusiasm. Again the resourceful department got busy and the Head forwarded an impressive letter to the great General. With elaborate phraseology and couched in the classical and cryptic "wen li" (the traditional dignified form of written Chinese) he politely asked the great General if it might not be possible to have his staff exercise a little more discrimination and tact in the technique of extermination of these unfortunate fellows so that the inexorable demands of scientific training might be more fully met.

A few days later the reply reached the department. Magnificent in red and gold and likewise couched in flowery and stilted language, the message of the great General was, in effect, as follows:

"Why, yes, certainly! I'll send them out to you, and you just kill them in any way that you wish!"

R. G. Agnew, B.A., D.D.S., F.I.C.A.

Note—We understand the Department offered the General a supply of chloroform.

The West African Ju-ju.

By OSKAR KLOTZ,
Department of Pathology and Bacteriology, University of Toronto.

The most powerful force guiding the native mind in West Africa is the ju-ju. The actions of every man, woman and child are dominated by fear, fear of the ju-ju. It is very difficult to give a definition of ju-ju, for words fail to describe the spirit of its meaning and the awe with which it is regarded. Some speak of the ju-ju religion, but it is not a religion; some speak of it as witchcraftery, but that is only part of its use; others refer to it as a charm, an image, a devil, a fetish, a spirit-power: a ju-ju is all of this and more.

The name ju-ju is thought to have come from the French word jou-jou, meaning a charm, a plaything, or a fetish. The Mandingos use the word grugu. The ju-ju is usually the thing which possesses the quality of a charm, but very commonly the term is used in the abstract, referring to the superhuman power which may be possessed by a charm, by an individual or by an unknown force. It is even used to mean the pagan religion itself. Lightning which has stunned a person may cause him to have ju-ju qualities. A twisted root having the appearance of a man possesses ju-ju powers. Various inanimate objects, such as shells, bones of man or animal, stones or hair may become infused with unusual powers by the touch or incantations of another ju-ju.

The West African ju-ju has many resemblances to our mascots, charms, fetishes and religious medals, while the spoken ju-jus are like incantations on the one hand and curses on the other. There is also much in common between the African ju-ju and the charms and superstitions of the Brazilian people. I well remember my surprise at the common practice of using charms to ward off misfortunes as I saw it among the caboclas of Brazil. And yet these people are devout Roman Catholics. Every farmer wards off the "evil eye" by placing the skull of an ox on the gate-post in front of his house. With this protection, no one possessing the power of the "evil eye" can by a glance destroy the crops or bring sickness into the family of the farmer. And from the innocent credulity of this enlightened white race, there has arisen the profession of witch doctors or feiticeiros. In Brazil, for a few milreis, you can hire a feiticeiro to cast a spell of ill-luck upon your sworn enemy; you can do even more,—that is, if you will add a few more milreis to your "dash,"—and have a fatal curse pronounced upon him. Lo and behold! your enemy dies, the curse has been effective and the witch doctor has decamped. Superstition and false faith are among the weaknesses of the human imagination. The human mind seeks explanations for all phenomena, and failing to find satisfaction in the few facts available, is content with the explanation evolved in the idle minds of "confidence men." An explanation sufficiently mysterious and involved is always more appealing than a simple one. This is true of all peoples and nationalities. For varieties of superstition, the Scotch and Irish are well equipped. Why, then, do we express such surprise and wonderment at the native African? The reason probably lies mainly in the fact that the African displays his beliefs and superstitions in peculiar actions and ceremonies; his surroundings are in such distinct contrast with the home environment, and his love of the grotesque, as seen in ceremonial dances and in the adornment of the ju-ju men and witches, is a rude shock to our "untrained" senses, untrained because we do not under-

stand the purposes of all these strange details.

It is very difficult to give an interpretation of the ju-ju belief in the English language, or, in fact, in any language save the Yoruba. We have not the words to express the sentiment underlying ju-ju thought, and if we use words and phrases which seem to paint a picture of the scene and the events, we fail to convey the sentiment of the action. Realizing thus the inadequacy of my vocabulary, I can do no more than illustrate by a few examples the variety of beliefs possessed by the Yoruba tribe.

The ju-jus of the Yorubas may be of the nature of charms; others are associated with places held to be sacred; others again are related to the powers of individuals such as witches or the high priests employed by the King; or again ju-ju powers are invoked by incantations or by certain mysterious phrases. Parallels to these customs are, of course, found in all parts of the world, differing only in detail. I remember a poor devil in Brazil who for much money purchased the prayer of St. John written in blood on a piece of dirty paper. The feiticeiro assured him that as long as he carried this prayer in his pocket, no harm could come to him, not even injury from revolver bullets. This credulous ass had a feudal enemy with whom he promptly got himself into trouble and started a revolver duel. They buried him next day.

Now, amongst the greatest enemies of the African, disease ranks in a high place. Malaria is always with them, dysentery stands close at hand and epidemics of a variety of fevers come and go with appalling regularity. These people are ill prepared to combat disease; of home remedies they have many, and while some of these are useful for minor ailments, they are quite useless in serious trouble. However, it is not the temper of man to stand by and admit his helplessness. Some comfort can be given the stricken patient and to the sympathetic relations by an appeal to the supernatural powers. And so we find a variety of ju-jus invoked to give aid to the unfortunate sick. For this purpose a person is called upon who has ability to deal with these superhuman powers, and in West Africa this person belongs to a distinct class, a kind of priesthood who not only are the vendors of charms and spiritual power, but also study the situation and then advise what shall be done. Their advice may have a medical leaning inasmuch as they are usually skilled herbalists; they may also suggest hydrotherapy or heliotherapy; and finally they may invoke the aid of "specific" charms. Special charms are made for special cases. They may consist of a mass of clay in which is kneaded powdered charcoal, ground peppers and a variety of animal substances. This mass is shaped to resemble some beast or man and may be applied to the patient in a variety of ways: sometimes it is simply placed in his room, or he may be allowed to touch it once; or the moist clay mass may be placed upon the part of the body suffering pain. In the latter case the presence of pepper in the clay serves as a counter-irritant and may actually have a beneficial effect. The ju-ju man always commands the patient to allow no interference with his instructions, for interference would rob the charm of its power.

The variety of ju-ju is endless. Seemingly any object may be endowed with ju-ju properties. Some wear armlets, neckbands, waist girdles, bangles and a great variety of trinkets which for the wearer have peculiar virtues as protectors. Every native city has its ju-ju market where one may purchase articles suited to his needs for protection against harm. This must produce a great sense of satisfaction in all who have faith in baubles and charms. And the faith of the native is very sincere; he will pay lavishly for the peculiar trinket which fascinates his desire. The children all receive the blessing of some particular charm, and those of

higher "social" rank are fairly weighed down with the variety of ju-jus.

The use of ju-jus is often determined by the interest of the chief of the community and his councillors. At Ifé, the centre of Yoruba tradition, the Oni is not only king of the district, but also high priest of the Yoruba tribe. His councillors are his intimate advisers, and some of these are ju-ju men of high order. Their influence pervades the entire district, and they have many subsidiary ju-ju representatives who keep in close touch with the people. These ju-ju men know all that is going on. No stranger comes to the city who has not been heralded to these "state officials." They serve as a secret service department. They know of the arrival of every new-born, of every death, of every pagan marriage. The very fact that the native administration has such close touch with all the events of the district has an important function in the care and control of the population. The king's messengers know where to put their finger on every emissary of the court, and within the period of an hour, detailed information respecting any subject of public or private affairs can be obtained in the king's council chamber. And so too the threat of the king's ill favor can be rapidly conveyed to the person or persons concerned. Such threats, having the weight of supernatural ju-ju authority, are much feared by the king's subjects, and so the king has a strong ally in his band of ju-ju men.

Amongst pagan people, then, there develops a tie between the state and "religion," such as we have known in European countries. Each element is helpful to the other. The fact that the native royal family gives support to the ju-ju cult lends great weight to the power exercised by these men, allowing them to demand heavy tribute for all favors given. The ju-ju men are crafty and are among the most intelligent individuals in the whole tribe. They know when to speak and when to maintain clam-like silence. They have good memories for events, places and persons. They are interested in everyone's activities and put themselves to trouble to draw strangers into conversation. They always dress in some distinguishing garb to denote their superior rank, and some of them acquire the added prestige of wealth gained from the donations of their satisfied customers.

With so strong a position, the ju-ju man finds little difficulty in leading the people into a make-believe world. No doubt each of them adds a mite to the over-stocked store of charms. With a good imagination he can easily set in motion a whole new train of thought for the visualization of the demon world. He can commercialize the fears of an innocent people, or the hopes of expectant parents. A mere verbal philosophy does not appease the spiritual craving of the uncultured tribe: there must always be material evidence. This evidence often strains the ingenuity of the ju-ju man, and yet on the success of his demonstration depends his future promotion. I encountered one ju-ju man in the interior who would have paid well for a small magnifying glass which on a bright day would ignite tinder or dry leaves. He saw possibilities of using it as a ju-ju. Another watching us do some urinalyses, marvelled at the blue litmus paper turn red with acid and then revert again to blue with alkali. This, too, had its possibilities. Wonder and curiosity concerning the unknown has always opened the way for wild speculation and deceit, in the metropolis as in the jungle.

The pagan people, like those of greater culture, feel a genuine awe for superhuman powers to whom reverence is regarded as justly due, and this is expressed conscientiously in the manner of life. Their lack of an orderly philosophy leads them to assume the existence of a variety of controlling powers which are beyond their comprehension and which are often believed to have the form of unseen man. Such powers are invested in certain spirits, not the departed spirits of their

ancestors, but the beings of an undefined spirit world which constantly keeps watch over the actions of men. Nevertheless the spirits of departed souls may also exhibit their interest in the doings of their earthly progeny by deeds which make for good or evil. To the native of West Africa, the tendency of all earthly affairs is for the good; wrong and evil and wickedness and misfortune are abnormal, the result of the successful entry of evil spirits into mundane affairs. The natural character of man is to do good; his perversion for wrong-doing is acquired only through the influence of devils guiding his actions. It is necessary, therefore, in their daily life to guard against the possibility of the devil or evil spirits gaining a foothold on their person or having an influence upon family, community or tribe. Their pagan belief, their religion, is centred upon the task of warding off evil. No help is sought from unseen supernatural powers to improve the status of good, of love, or of beauty. These remain in the sacred state in which the God of creation placed them. None can do better than the good which is his heritage; but there is much need for protecting this human bequest.

Every pursuit of a native tribe is beset with dangers imposed upon it by the particular evil spirit. Those engaged in fishing for example must by some means combat the evil spirit which makes them unsuccessful in gaining a good catch, which destroys fish, which hastens storms to overtake them before they reach land, and which does a great many other things to destroy their happiness in their occupation. So, too, there is an evil spirit confusing the hunter, the farmer, the potter, the weaver, the expectant mother, the chieftain and the children. Other evil spirits molest the small communities, destroying their houses, poisoning their wells or devastating their plantations. These must all be warded off or appeased in the fashion which seems best for the occasion. They cannot all be dealt with in the same manner, for, according to the season and the moon, special rites and ceremonies must be undertaken. Rufus, my hunter, told me that he could not underake to kill any large animal, for should he do so the devil would bring vengeance upon his pregnant wife. He must wait until the child was born, and after that he must offer his first trophy to his spirit watchman. Inasmuch as there are so many evil spirits, each awaiting his opportunity to pounce upon an erring human, the native finds it wise to seek the counsel of wiser minds. And this is when the ju-ju man plays his part in controlling the actions of young and old. A wise ju-ju man exercises great power. He must be discreet, tactful and familiar with all the peculiarities of his flock. The best of them are stolid, uncommunicative and of good bearing, winning the confidence of the people without, however, allowing them to become familiar with their way of living. The ju-ju man pretends to possess profound secrets, and at times of murder and civil insurrection, he does. Not only is he the human oracle from whom may be obtained advice and instruction, but he is often also the active participant who performs the necessary rites to allay the wrath of the gods. None is more pleased than the ju-ju when misfortune or disaster falls upon a wealthy community, for then he may stage great and varied festivals, ju-ju dances and ceremonies, while, then, too, he reaps a great harvest for his private coffers by the sale of protecting images. The native town of Asamenkase (Gold Coast) when stricken with yellow fever, called for a ju-ju man from Dahomey. The fee he asked for his visit was £500. He actually received £250 before he was hounded from the town for failing in his promises. Even the ju-ju man must "deliver the goods" or fall into disrepute.

It is interesting to note that in all forms of religion, a great deal of the "vis a tergo" which drives the people to follow the straight and narrow path of their creed lies in fear. Fear is

a directing force in the lives of all people, people in all walks of life, from the characters of the underworld to the idolized saints. True it is that the nature of the fear differs widely in different individuals. One fears physical pain; another fears disgrace or the hell of the afterworld; another the lowering of his station in life or his prestige in the eyes of his family or friends —there are a thousand and one fears or things to be afraid of, and each of us has his own conscious or subconscious fear, which is largely the guiding hand of his destinies.

And so, too, our untutored people of Africa, whose ability to reason is undeveloped and whose opportunity for learning to differentiate between the truth and the ridiculous has been denied them, are governed by fears arising in the mysterious workings of nature. Fears are constantly in the background of their lives, but none wish to admit them. Such an admission would indicate physical weakness, so it is better to ascribe their fears to superhuman forces, spirits, gods or devils. There seems to be a devil lurking behind every tree, around every corner, above every roof-top, and below all water,—devils great and small, devils which are only mischievous, devils which are annoying, and devils which are actually harmful. For the native of Africa, the world is filled with innumerable devils which are attempting to bring misfortune and distress, and against which it is man's greatest task to wage constant opposition. Many methods may be invoked, but the best concerns itself with appeasing the wrath of the evil ones by bribery or gifts.

To this end everyone learns the more common rites necessary to keep the evil spirits in pleasant humor. I presume that it is the interest which the native takes in food and drink which makes him regard these necessaries as highly efficacious in devil worship. Many ju-ju altars have a good supply of food; other sacred places likewise have food, often of a special kind. Medicine pots are erected on posts with three pronged limbs and the pots are filled with a variety of ingredients, leaves, nuts, animal bones and so forth. There are just as many ju-ju charms as the priests' minds can conceive. No doubt the ju-ju brings comfort to many, particularly those who are wrought up and nervous and those who have exaggerated a danger in their own minds. Faith in the efficacy of the charm must help to allay such unnecessary fears.

Very often the entrance to the house has a small decoration above the doorframe. This decoration may consist of bits of rags rolled, knotted or tied into curious shapes; bits of bone, white, blackened or coloured; or again, masses of twigs tied together with human hair. Each of these objects has a meaning for good or bad. Some are placed there by the householder, others are put there by his enemy. Ju-jus for these and all other uses may be bought in the market-place, where there is always a section reserved for the sale of charms. This section is one of the most interesting parts of the market in a West African town, for one may see there all kinds of strange objects which are claimed to have peculiar powers against evil spirits.

Closely allied to the belief in the ju-ju charm is belief in the powers of certain individuals or groups of individuals, who stand at the head of peculiar cults and secret societies: the smallpox cult, the lighting cult, the alligator society, the leopard society and many others. Each of these is under the guidance of a few men who serve as high priests, whose word is law and who have the right to impose the death sentence if the infraction of their regulations demands it. Very little can be learned from the natives regarding these societies. Their fear of serious punishment for divulging any information concerning the purposes of the society, its officers, its enemies, etc., is very great. Only once did I hear a native tell something about the small-

pox society. He was a native doctor who had been educated in Edinburgh and who was practising in Lagos. He related that a smallpox society had existed in Ije-obode. The government had never been able to make any headway in vaccination in this town, where there were recurrent epidemics of smallpox. He had been in Ije-obode for some time and, wishing to know more about this cult, used all his influence to become a member of the group, succeeding after a time. He found that the head of the organization knew something about smallpox and taught the people to avoid vaccination or, if they were forcibly vaccinated, to wash and treat the scarified area with certain decoctions. On the other hand, this priest, deeply pock-marked himself, collected pus from active cases of smallpox, stored it in small vials and kept it in a cool, dark place. If in his opinion the community had been peaceful for too long, or if some political or other problem gave rise to dissension, he would undertake to get a smallpox epidemic under way. At other times he spoke as the oracle of the gods, and would then prophecy some calamity. Having the equipment for such a calamity at hand gave him great power. The raging of a smallpox epidemic in a native town is worth riches to the ju-ju men, who act both as herbalists and charm doctors. This doctor told us that he was able to give the government sufficient information to enable it to disorganize this society.

There are ju-ju men who can give absent treatment. Many a man has travelled miles to the house of the ju-ju man to tell him of the agonies of a sick wife or child. The whole story is told, how the illness began, where it seems to be located, how it progressed, and the failure to obtain relief by the use of vegetable infusions and other home remedies. If the ju-ju man accepts the obligation of treating the case, he may never go to see the patient, but will instead repeat in monotones certain phrases not unlike the incantations of our Northwest Indians. Or he may make a ju-ju charm which is carried back to the sick person to touch and gaze upon. Ju-ju men who possess such powers of absent treatment are in great favour.

Nearly all ju-ju men wear silver rings, one on the index finger of each hand. The ring on the right hand has the property of causing sleep in the person who is touched by this hand. This sleep, it is claimed, is so deep that the patient is anaesthetic and may be incised without pain. The ring on the left hand is endowed with power to wake him again. I have never seen these charms in use, but was told in good faith by several natives, including the native doctor at Lagos, that this was true!

However, is all of this so astonishing? We can probably match the favors of the African ju-ju, case by case, with still more remarkable examples of modern superstition. Only recently, Sir D'Arcy Power commented upon this as follows: "But this is no matter of surprise, for in 1925 Mr. Edward Lovett published a book which he called Magic in Modern London. He showed from personal knowledge that nearly all the old charms, mascots and superstitions are in active use amongst us at the present day. It is, for instance, still possible to buy a wind by the purchase of a knotted string; to save oneself from drowning by carrying a caul —cauls went to a premium during the war—or to loosen cramps by keeping a fossil shark's tooth in the trouser pocket. Indeed nearly every motor car has its mascot 'for luck' and the plaques of St. Christopher—the patron saint of travellers—are innumerable. Superstition, therefore, is not confined to the uneducated."

In Memoriam

DR. GORDON HALL RUTHERFORD.

DR. GORDON HALL RUTHERFORD.

Dr. Gordon (Hiker) Rutherford, while attending his patients in his office in Chatham, apparently in perfect health, was suddenly stricken and succumbed within one hour, the evening of January 4, 1932.

He was born in Blenheim, Ontario, on September 1, 1900. There he received his primary education. At the age of sixteen he enlisted as a Gunner in the Canadian Artillery, and served Overseas until the termination of hostilities. On return, his interrupted primary education was continued at Woodstock College. He graduated from there with the Governor-General's Prize for Proficiency. He entered the study of medicine at 'Varsity in the class of '27.

As an undergraduate in medicine, his life was one of incessant activity, which manifested itself in every phase of student life. He was engaged in many branches of sport, and was a member of the Senior Medical Rugby team, Mulock Cup winners in 1925. His magnetic personality and his admirable character made themselves felt to best advantage in the government of the medical undergraduates, and culminated with his election to the Presidency of the Medical Society, and Board of Stewards of Hart House, in which capacity he made himself loved and respected by all. He was a most esteemed member of the Phi Chi Fraternity.

On graduation, he interned in St. Francis' Hospital, New York City, later married, and began practising in Chatham. He took a very active part in community life, especially Amateur Athletics. He was the Medical Officer of the Kent Regiment. He is mourned by all who knew him.

Bright's Disease.

I. CLASSIFICATION, AETIOLOGY, AND PATHOLOGY.

By F. S. BRIEN, B.A., '33; W. J. HENDRY, B.A., '33; R. B. KERR, B.A., '33.

It is the purpose of the authors to present a series of three articles on the general subject of Bright's Disease. The current article will deal with the classification, aetiology and pathology of the lesions found in this group of conditions, while subsequent discussions will cover their symptomatology, diagnosis, laboratory and clinical, and treatment.

ANATOMY AND PHYSIOLOGY.

The reader will be familiar with the histology of the unit of kidney structure, the nephron; however, there is one detail of the blood supply of that structure which deserves special mention. The main blood supply to the tubules of the kidney, the cortical labyrinth, is from a group of capillaries formed by the breaking up of the efferent arteriole of the glomerulus. Hence, the blood which supplies the tubules has previously passed through the glomeruli.

For a concise and complete account of the physiology of the kidney, it is recommended that the reader consult an article by Miss J. C. Gray in the U. of T. Medical Journal of April, 1931. The present authors feel, however, that enough stress has not been laid on the functions of the glomerular capsule. Through this membrane all the fluids filtered by the glomerulus must pass. Urea, glucose, and sodium chloride may pass readily through this epithelial investment, while the much larger protein molecules are held back. The permeability of the capsule is very delicately balanced and may be disturbed by processes which produce no demonstrable histological change. Hence, one must realize that albumin may appear in the urine without any visible alteration in any part of the nephron.

This semi-permeability of the capsular membrane has an extremely important bearing on the mode of action of bacterial toxins on the kidney structure, in as much as these toxins are of protein nature. Further reference to this subject will be made under the heading of aetiology.

DEFINITION.

The modern concept of Bright's Disease includes those lesions of the kidney which may be spoken of as "Medical Kidneys." They are non-suppurative bilateral lesions which may be recognized by abnormal urinary findings and clinical signs such as oedema and high blood pressure.

CLASSIFICATION.

Since Richard Bright first published his epoch-making study of the condition which bears his name, the term Bright's Disease has been extended to include lesions other than those which he described. Until recently the pathological and clinical concepts of the findings have been in a state of chaos.

The following classification has been suggested to correlate clinical and pathological findings in the varied lesions seen in Bright's Disease.

A. Nephrosis—degenerative lesions.

B. Nephritis—inflammatory lesions.
   (1) Acute.
   (2) Subacute.
   (3) Chronic.
   (4) Malignant or Combined form —in this type there is a combined lesion of the kidney parenchyma and an arteriolar lesion which tends to occlude these minute vessels.

While it is convenient to classify nephritis into the above more or less

definite types, it must be recognized that there is a considerable blending and overlapping of the various stages in any one particular case.

It should be stressed also that in any stage of the nephritic process, either the glomeruli, the interstitial tissues, or the vascular structures may be most prominently involved. Hence, on this basis we may have a classification of any of the above stages into:

(1) Glomerulo-nephritis.
(2) Interstitial nephritis.
(3) Perivascular nephritis.

AETIOLOGY.

Nephrosis.—The degenerative processes in the renal tubules, which constitute nephrosis, are the result of the action of toxic substances. The latter are usually bacterial in origin, although chemical poisons (e.g., mercuric chloride), foreign proteins, various venoms and plant poisons will also produce the same effect.

Nephritis.—It is believed that the primary reaction in nephritis is an inflammatory change in the gomeruli. This appears to be bacterial in origin, due to the action of their toxins on the kidney structure. Gray has recently advanced an interesting explanation of the tendency for the reaction to centre in the glomeruli and for the failure to recover living organisms from the lesions.

He suggests that, due to the semi-permeability of the capsular membrane, the toxins, which are in themselves proteins, do not pass through this membrane, as mentioned above. The fluids readily pass through the glomerular capsule, causing a concentration of these toxins. This results in inflammatory changes in the glomerulus. Soon the glomerular membrane is damaged by the toxins, thus allowing their passage into the tubule, with subsequent degeneration of the epithelial cells of this structure.

Because of the definite association between certain bacterial infections in other parts of the body and nephritis, it is believed that the causative toxins arise from these sources. The onset of the disease is very frequently associated with some upper respiratory infection, such as abscessed teeth, tonsillitis, sinusitis, and infections of the bronchial tree. The streptococcus plays an important role in the causation of these conditions. Scarlet fever is also a frequent precursor of nephritis. Strength is lent to this view by the recovery of cases of nephritis immediately following the removal of such foci of infection.

PATHOLOGY.

A. Nephrosis.—The term nephrosis was introduced by Friedrich Mueller in 1905, to indicate a degeneration of the renal tubules. This lesion may be accompanied by a slight inflammatory reaction of the gromeruli. The following stages may be recognized in the pathological process of nephrosis:

(1) Cloudy swelling.
(2) Extensive cellular changes.
(3) Repair.

Macroscopic appearance.—In the first stage, the kidney is congested and swollen. On cut section the cortex is thick and bulges above the surrounding tissues. The capsule strips freely. As the process progresses the kidney becomes pale and yellowish, with yellow streaks through the cortex. As the stage of repair is reached the organ becomes reduced to normal size.

Microscopic appearance.—The epithelial cells of the proximal convoluted tubules, chiefly, show typical degenerative change. The first stage is characterized by cloudy swelling of these cells. Hyaline droplet degeneration appears as the condition progresses, to be followed by the appearance in the cells of droplets of a lipoid material, which stain with Sudan III., or Scharlach R stains. Amyloid deposits may be seen toward the basement membrane of the cells of the tubules, in the media and intima of the arterioles, and in the glomeruli around the capillaries beneath the epithelial membrane. This condition may result in a fatty or hyaline change in the affected cells. In severe

forms of nephrosis, cellular debris may be found in the widened lumina of the degenerated tubules. Upon recovery from the condition, the kidney returns to normal so that no evidence of the lesion can be detected. There may be some slight inflammatory infiltration of the renal structures in nephrosis, as well as the above described degenerations.

B. Nephritis.—In nephritis a non-suppurative inflammatory reaction is seen in the kidney. Characteristically there is involvement of glomeruli, interstitial tissue, and vascular channels. As previously indicated, any one of these areas may be involved to a greater extent than the others, which, however, invariably show some degree of pathological change. The site of maximum change can be determined only by microscopic examination of the tissue. As already mentioned, the blood supply to the tubules first passes through the glomerulus. For this reason when the latter is involved in an inflammatory process, the nutrition of the tubule suffers and degeneration takes place. Hence every nephritis has an accompanying nephrosis.

(1) Acute Nephritis.

Macroscopic appearance.—The kidney is slightly larger than normal; the swelling being due to inflammation—oedema and congestion, as well as, to some extent, swelling of the parenchymatous cells themselves. The renal capsule is tense and stretched to such an extent that when it is incised the kidney substance bulges. The capsule is thin and strips easily. The underlying tissue is congested and is dotted with punctate areas of haemorrhage, which takes place into the nephron itself and not into the interstitial tissues. On section the cortex is seen to be thicker than normal and to bulge above the level of the medulla. The latter may show some congestion, but no inflammatory reaction is discernible as a rule.

Microscopic appearance. — The glomeruli may all be attacked, but not necessarily so, and there appears an infiltration of lymphocytes and some polymorphonuclear leucocytes, which appear in numbers in the free capsular space, so being observed in the urine, even if the reaction is non-suppurative in character. Other areas of cellular infiltration are seen around Bowman's capsule, in the interstitial tissues, and about vascular channels. The glomeruli are the seat of marked proliferative change. The capillary endothelium proliferates to a marked degree, while the epithelium of the capillary tuft does so to a less extent. Intracapillary masses of hyaline fibres are formed. Thus the vascularity of the glomerulus is markedly reduced; hence some slight degree of tubular degeneration is to be expected in this stage of the disease.

In vegetative endocarditis, as a result of embolism, some glomeruli may be infarcted, so giving rise to an acute focal glomerulo-nephritis.

(2) Subacute Nephritis.

Macroscopic appearance.—The kidney is enlarged in this stage, often to a greater extent than that seen in the acute phase. The capsule is thin and strips readily, exposing a surface which is smooth and very pale, almost white, thus the term "the large white kidney" or "the hog-back kidney." The cut surface is very pale and reveals a marked swelling of the cortex. The organ is soft in consistency. The swelling and pale colour of the organ are due to the deposition of lipoid materials in, and swelling of, the cells of the tubules. The macroscopic appearance of the kidney at this stage resembles to a considerable extent the picture presented by the organ in nephrosis.

Microscopic appearance. — The glomeruli are enlarged and avascular. The entire structure is converted into a hyaline mass by an extension of the process observed in the acute phase. The capsular space may contain red blood cells, desquamated epithelium and fibrin. A striking proliferation of the capsular epithelium has occurred, often filling the capsular space. Frequently this cellular mass assumes a

crescentic form, and hence is referred to as an "epithelial crescent." The neighbouring tubules show cloudy swelling, fatty degeneration, or severe necrosis. Many of the injured epithelial cells are cast off into the tubules to form epithelial casts.

(3) Chronic Nephritis.

Macroscopic appearance.—The kidney in this stage is small, contracted and granular. It is covered by a capsule which is thicker than normal. This is so firmly adherent, that portions of kidney substance are torn away when the capsule is stripped. This is a result of the involvement in the inflammatory process of the vessels which enter the kidney from the capsule. They form fibrous tissue and bind the capsule firmly to the kidney substance.

If cardiac disease is present, "the small red kidney" is seen due to chronic passive congestion. In the absence of a heart lesion the "small white kidney" is observed. Both types of kidney are similar except that one is complicated by cardiac disease. Often arterial disease is also present; this constitutes the so-called "Triple Alliance" —cardio-vascular-renal disease.

The cut surface shows a fairly sharp line of demarcation between cortex and medulla. The cortex is small and narrowed, but the shrinkage is irregular in distribution, thus accounting for the granular appearance. The medulla may be slightly narrowed. Small cystic spaces filled with a clear fluid are often present in the cortex—retention cysts.

Microscopic appearance.—Fibrosis is marked in this condition, in some cases completely obliterating the normal architecture of the kidney. The glomerular lesions have progressed until nothing but a hyaline mass remains. The glomerular spaces may be completely obliterated. Neighbouring tubules are shrunken from the markedly increased fibrosis, which surrounds them, and may show the most advanced degrees of atrophy. The blood vessels show fibrotic changes in their immediate neighbourhood and a thickening of the inner coat with a consequent narrowing of the lumen is to be observed. A curious feature of this stage of the disease is that many glomeruli appear to be in a fair state of preservation and around them there may be areas of regenerating tubules.

Acute exacerbations of nephritis may occur in chronic nephritis, and engrafted upon the picture just described, one may find the characteristics of the acute stage of the inflammatory process.

(4) Malignant or Combined Form of Nephritis.

Macroscopic appearance.—The kidney is small and shrunken. The capsule is adherent to the surface of the kidney which is finely granular and mottled. Small cysts may be seen on the surface of the organ. The cut surface shows the irregular atrophy, char-

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acteristic of chronic nephritis. However, the small blood vessels, particularly those at the boundary between the pyramids and cortex, present greatly thickened walls and gape widely open.

Microscopic appearance.—The characteristic lesion is a peculiar uniform hyaline thickening of the sub-endothelial layer of the intima of the small arterioles, which may completely occlude their lumina. The earliest change observable, however, is a dilatation of the afferent arteriole at the point of junction with the glomerulus and the capillaries springing from it. The interlobular, medium-sized arteries of the kidney show a thickening of their inner coats and a reduplication of their inner elastic laminae.

There is another type of arterial degeneration seen in the kidney, particularly in the renal artery and its larger branches. Their lumina may be diminished or even occluded, thus giving rise to a slow process of atrophy with the production of a lesion, not unlike a white infarct. The vessels so involved are not in the parenchymatous tissue. This is an example of true arteriosclerotic atrophy of the kidney. Klotz does not believe that this lesion should be included in the condition of Bright's Disease, although some other authors class it as such.

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The authors wish to express their appreciation to Dr. W. L. Robinson, of the Department of Pathology, for his criticism and suggestions in the preparation of this article.

(To be continued)

National Health Insurance.

By JOHN L. CATHIE, '37.

The subject of National Health Insurance has loomed large in the attention of the members of the medical profession in Canada for some time, and it is a widespread idea that, sooner or later, some system of industrial health insurance will be introduced in this country.

Coming from Scotland, where a national scheme of this kind has been in successful operation for over twenty years, I feel it may be of some interest to indicate just how this Act affects the general practitioner.

Under the provisions of the National Health Insurance Act of Great Britain, all employed persons earning under £250 (two hundred and fifty pounds sterling), per annum, are required to take part in a scheme of State Insurance for health purposes. Fifteen million persons are affected, and the total cost per capita works out at about thirteen shillings each year. The State pays one-seventh of the cost for men and one-fifth for women; the balance is distributed between employer and employee. Employer and employee pay weekly contributions, the State adds the grant from the Public Exchequer, and the insured person becomes entitled to certain benefits, the principal being medical services, viz.: (1) Medical attendance and treatment by a general practitioner; (2) proper and sufficient medicines and a limited number of medical and surgical appliances; (3) periodical cash payments during sickness and disablement; (4) maternity benefit in a lump sum payment on

birth of a child, increasing according to the number of children in a family.

To secure these benefits, a record of illness and a certificate of incapacity must be furnished by the "panel" doctor.

For the purpose of administration, medical areas, corresponding to counties or townships, are located throughout the country, and in each area are committees authorized by the Minister of Health and his advisors, as follows:

(a) Local Medical Committee, composed of all doctors, insurance or otherwise, to look after the interests of the Medical Profession in general.

(b) Local Medical Insurance Committee formed from representatives of "Panel" doctors, druggists, and insured residents, and responsible for the maintenance of an adequate medical service to insured persons in their area;

(c) Special Board of Enquiry, consisting of a barrister, two doctors, and a neutral president, to handle all compaints regarding infractions of the regulations, monetary adjustments, etc.

Every qualified practitioner is entitled to treat insured persons, and although he can, on one month's notice, withdraw from "Panel," he cannot be dismissed by even the Minister of Health unless a complaint, sufficiently serious to warrant his exclusion, is sustained by the Special Board of Enquiry. Removal from the "Panel" does not affect his standing as a licensed practitioner, unless the charge concerns a criminal offence over which the Licensing Board has jurisdiction.

Insurance doctors have the right to reject any insured person, and insured persons have the unassailable right to choose any doctor or alter their choice, but all "Panel" doctors are responsible for the insured person's medical attendance, and if the latter's own doctor is not available, any insurance physician must substitute, on demand.

In allocating the number of patients, regard is had for the population of the medical area. The average is 2,500, but the absolute limit is 3,000. In sparsely populated districts the number may be much less.

Remuneration is apportioned by means of two expenditure "pools," the first — a Central Pool — is an estimated amount of the total cost of medical services for the whole country, including hospitals, supplies and remunerations; and the second — the Local Pool — an estimated amount sufficient to cover total cost for each local area.

From this Local Pool certain deductions are first made — (a) one-quarter for a Practitioners' Fund to provide remuneration for special services rendered — operations, anaesthetics, emergency calls and night calls; (b) a fund for Panel Committees expenses; (c) travel funds for practitioners in rural areas. The remainder is distributed to the "Panel" doctors, quarterly, on a units of credit system. Say one doctor had 1,000 units of credit, i.e., 1,000 recognized, insured persons on the list, the balance of Local Pool was £3,500, and the number of local credits 30,000 units, then that doctor receives £116 13s. 4d. as his share, or about $583.

As to the Panel doctor's duties, he is required to give only what is estimated to be within the scope of the average general practitioner of his area. Specialists' care or extra calls are chargeable to the patient. He is, however, obliged to refer his patient to another whom he considers more fit to take charge of a case if he considers his own experience or resources inadequate. In this event the Local Committee decides on the monetary adjustment. It is left to the judgment of the Panel doctor as to what he considers the proper medical treatment and medicines required, and in the earlier years of the system's operation many fine points of controversy arose as to just what "proper medical treatment" constituted. Dental services were ruled out almost immediately as a benefit to which the insured person could lay claim. It is interesting to note, that medical attendance can be claimed by patients suffering from diseases or afflictions intensified by their

own addiction to alcoholic or other excess, but, should a doctor recommend a cessation of the excess, and same is not carried out, the patient's name can be struck off the list by the Panel Committee, on receipt of a certificate to that effect from the physician. From then on, any service rendered to that patient becomes chargeable. Apart from this angle of the case in question, chargeable services have been listed systematically, as the years pass.

Medicines are dispensed by Panel druggists, who are paid a flat rate per type of prescription through the Local Disbursement Fund. The latter fund also takes care of the rural doctor who must dispense his own prescriptions.

Cash payments for disability are paid to the insured person on presentation of a medical certificate, and cease when the doctor submits a certificate of person's fitness to resume work. It is a peculiar feature of this cash payment that most are paid through Approved Societies. So prevalent were these Sick Benefit Societies when the N.H.I.A. was introduced, that the Government was forced to recognize them; consequently, Approved Societies, within the meaning of the Act, handle the Disability Benefits. Persons who are, however, not members of any society may obtain their grants through their local Post Office.

Ordinary hospital treatment is given and in institutions where an adequate medical and surgical staff is maintained, the insured person must accept same, the attendance of his doctor being regarded as an extra service. Special Tuberculosis Clinics and Dispensaries are located strategically under the provisions of the Act, and skilled treatment given for all stages of the disease.

The National Health Insurance Act, protecting fifteen million people, has been in force since 1911, and its success is reflected in the satisfaction of general practitioners, and a betterment of health conditions throughout the land.

It is a well-known fact that the wage-earner who is not a member of any sick benefit society usually does not apply for medical treatment for his own illness until some disease actually incapacitates him for work. Thus, he might suffer for years without medical aid, and even find, when the ravages of his affliction render the enlistment of a physician's services compulsory, that it was too late to affect a cure, or indeed to save his life. Under the N.H.I. Act the insured worker has a doctor, in the payment of whose services he is sharing in any case, who will give him skilled treatment for the smallest cut or burn, will dispense any needed medicine or surgical appliance, and secure for him a cash payment should his injury or sickness necessitate a withdrawal from his accustomed avocation.

Proper care of maternity cases removes the dangers due to carelessness and even neglect.

In considering the advisability of a State Insurance scheme in Canada, we have the decided advantage of being able to study the system found in actual operation in the Old Land. From some angles, at least, it would seem advisable to look with favour on any reasonable State Insurance scheme which may be advanced.

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Acute Appendicitis.

By J. W. BRENNAN, '32.

I. PATHOLOGY.

Historical Sketch.

Our knowledge of the pathological conditions found in the appendix is a result of investigation which took place almost exclusively in the nineteenth century. Reference was made to disease in its structure previous to this, but the significance of the lesion was not appreciated and the finding was regarded as a curiosity.

The first reference to disease of the vermiform appendix of which we have record was made by Heister in 1755 in an autopsy record, where he described a gangrenous appendix. Mestivier in 1759 was the first individual to report a case of acute appendicitis during life. Louyer Villermay, a Frenchman, in 1824 described in detail the morbid anatomy of two cases. He expressed the fact that inflammatory processes in the appendix develop and progress rapidly. Melier, another Frenchman, in 1827 described five cases. He recognized the possibility of fecal concretions as a casual factor of acute appendicitis. Dupuytren opposed the idea that inflammatory processes in the right iliac fossa developed from morbid conditions in the appendix. He stated that the reason for their presence lay in the structure of the ileocecal valve and in the caecum, and coined the term perityphlitis; due to his prominence as a surgeon his theory became established and remained dominant for almost half a century.

In 1886 Reginald Fitz, Professor of Pathology at Harvard Medical School, presented his paper on "Perforating Inflammations of the Vermiform Appendix." The most important point which Fitz grasped was that the various abdominal disorders known as perityphlitis, typhlitis, caecitis, etc., were nothing more than different manifestations of a morbid process originating in the vermiform appendix. He introduced the term appendicitis and spoke emphatically upon the necessity for prompt surgical intervention in the treatment of this condition.

In the United States the first operation on the appendix was performed by J. Hall in 1886. The first occasion on which an unhesitating diagnosis was made before operation occurred in 1887, when Dr. H. B. Sands was the surgeon.

Anatomy.

The appendix in man is a vestigial organ related to the large caecum of herbiverous animals. In foetal life it forms the end of a pouch, the caecum, which buds from the large intestine. Towards birth the caecum enlarges without a corresponding growth of the appendix, a disproportion which continues to increase up to adolescence.

In structure the appendix resembles the large bowel. It is a hollow tube representing a crypt. It is unusually rich in lymphoid tissue, some of which may be found in the submucosa. The length of the appendix varies greatly from a mere stump to as long as eight or nine inches, averaging from three to four inches. The organ possesses a mesentery in the free margin of which is found the appendicular artery, a branch of the iliocecal appearing from beneath the iliocecal junction. In contrast to the intestinal arteries supplying jejunum and ileum, which divide and anastomose forming arterial arcades, the branches of the appendicular artery have a poor anastomosis, a fact which probably has great influence upon inflammatory processes arising in its structure.

The position of the appendix varies greatly, but as a rule it arises from the inner and posterior aspect of the caecum about one inch from the iliocecal junction. However, this position is influ-

enced greatly by variations from the normal n the descent of the caecum. If the appendix becomes fixed before the caecum has completely descended, the latter overlies it, and a retrocaecal appendix is the result. The caecum may fail to descend, and then the appendix remains high in the right side of the abdomen in the neighbourhood of the gall bladder. When the caecum is quite mobile, the appendix may lie to the left of the mid-line or within the pelvis. By following the longitudinal bands in the colon downward it is noted that they converge at the root of the appendix. This procedure may be used to locate the appendix at operation.

The function of the appendix has been a matter for great speculation. Many functions have been ascribed to it, but the experimental physiologist has not definitely demonstrated any. In herbiverous animals, where this organ is quite large, it probably serves an important role in the digestion of cellulose.

Aetiology.

The results of the bacteriological examination of the contents of inflamed appendices vary. It is not the mixed flora of the intestinal content. Bacillus coli is usually the organism which predominates, but that whether the bacillus coli can produce the severe forms of appendicitis is doubted by many pathologists. Boyd states that it is probable that many of the severe and virulent infections associated with rapidly-spreading peritonitis are due to a streptococcus. Other pathologists assign important roles to anaerobic organisms. The mixed flora present in intestinal contents renders it quite difficult to arrive at definite conclusions regarding a causative organism.

There are four possible routes of infection:

1. Haematogenous.
2. Enterogenous.
3. By lymphatics.
4. By direct extension.

It seems probable that the appendix can be infected by the haematogenous route because the disease not infrequently occurs simultaneously with, or a day or two after, an attack of acute tonsillitis. However, the enterogenous origin is the oldest view and the most logical. Aschoff believes that the inflammatory process begins in the crypts present in the mucosa where mechanical conditions favour the growth and action of bacteria. At the beginning of the attack there is a small break in the mucous membrane covered with a small mass or plug of fibrin and polymorphonuclear leucocytes. The inflamed area leading from this primary lesion is usually wedge-shaped, the apex of the wedge being directed towards the lumen. This may be complicated by minute intramural abscesses which can, by increasing in size, dissect the mucosa or perforate externally or internally.

The couse is often quite elusive. When the peristalic action of the appendix is normal the mucofecal contents are constantly being discharged. Retention is assured with insufficient peristalsis; stasis in the caecum due to chronic constipation, irregular position, excessive length or the presence of fecal concretions. In all these conditions the contents of the appendix necessarily stagnate, and this always causes an increase in the number and virulence of the bacteria present. It is possible that some of the symptoms thought to be due to an acute inflammatory process in the appendix may in reality be caused by muscular spasm or distension, due to a fecolith acting as a ball valve.

Pathology.

Inflammatory processes in the appendix may terminate in one of three ways: resolution with return to normal, healing with scar formation, or tissue distruction with suppuration, ulceration, or gangrene. The inflammatory process has been classified in many ways according to stages. It is very difficult to correlate clinical symptoms with pathological changes, and clinically it is impossible to differentiate the various forms, as they are merely stages

or degrees in a continuous process which may pass rapidly from one into the other.

1. Acute Catarrhal Appendicitis.— In this type the inflammatory process involves for the most part the mucous membrane, which becomes swollen, oedematous, and infected. The appendix is slightly thickened, and the subperitoneal vessels are dilated and tortuous. Microscopic section reveals the tissues to be infiltrated with leucocytes while the lymph follicles are more or less swollen. The mucous membrane is not always ulcerated, and healing may take place with no residual changes, but it usually results in stenosis after ulceration, or kinking due to peritoneal adhesions — residual lesions which predispose to further attacks.

2. Acute Diffuse Appendicitis. — Where the inflammatory process very early extends beyond the mucosa, and there is a general involvement of all coats, the condition of acute diffuse appendicitis is said to exist. The appendix shows a noticeable increase in all its dimensions, and may be twice its usual length and as thick as the index finger, the tip being slightly clubbed. Its colour is a diffuse bright red or dark mahogany mottled with subserous extravasations of blood, and often presenting light yellowish or greenish yellow areas due to localized foci or suppuration or necrosis. The mucous membrane becomes granular, and frequently there are more or less extensive erosions, which vary from slight superficial ulceration to extensive loss of tissue, compressing a large portion of the mucosa involving the submucosa and muscular coats and leading to perforation.

Microscopically all coats are involved in the inflammatory process. They are swollen, oedematous, congested and infiltrated with polymorphonuclear leucocytes. Due to the great swelling of the interstitial tissue, the glands are compressed and show degenerative changes. The mucous membrane over a considerable area of the lumen may be obliterated and the exposed surface covered with an exudate of polymorphonuclear leucocytes and fibrin. Haemorrhages are frequently noted in the mucosa and submucosa.

Acute diffuse appendicitis may undergo repair, but a complete restitution of structure is probably very rare. As a rule various deformities remain, such as angulations, twists, and what are perhaps most important, are irregularities in the canal — scars and strictures. Where the inflammatory process is not arrested at this stage, it may progress to gangrene or perforation. The increased tension causes an increased passive congestion of the appendicular wall, and this congestion may cause acute interference with the blood supply. This interference with blood supply is greatly increased if there is a pre-existing twist or angulation, or pressure on the inflamed and oedematous mucosa by a concretion. Vascular thrombosis occurs, and necrosis and putrefaction of the tissues takes place. If thrombosis affects the main artery of the appendix the whole organ may become gangrenous. More often a localized area of gangrene results near the tip due to thrombo-arteritis of one of the smaller arteries as a result of the surrounding acute inflammatory process.

Perforation may take place in any variety of appendicitis and at any stage of the attack. A purulent inflammation confined to mucosa and submucosa may extend outward by way of blood vessels and lymphatics in the hiatuses of the musculature, and cause an acute peritoneal reaction or even perforation without involvement of the musculature. One of the most important causes of rupture is necrosis of the tissues induced by the presence of concretions, the deleterious effect of which is only brought into play by the swelling of the tissues in the acute inflammatory process. Or it may follow erosion. Here the procedure is gradual, the various layers giving way as the degenerative process extends outward.

The favourite place for perforation is at or near the tip. In such a gradual perforation fibrinous exudate and omental adhesions often wall off the region of penetration, or at least localize the inflammatory process resulting in a periappendicular abscess.

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(To be Continued).

The Physiology and Pharmacology of Iron.

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The amount of iron required daily in the body to maintain haemoglobin within normal limits, is in the neighbourhood of one or two milligrams. The quantity excreted is practically the same. This amount is obtained from a normal diet or from almost any form of iron which may be administered per os. Recent investigations have demonstrated that ferrous salts are the most active therapeutically. The evidence presented indicates that when other salts are administered orally, only that portion which is reduced to the ferrous state ever becomes available for absorption and haemoglobin formation.

Some investigations by Lintzel have produced very interesting results. In one experiment, a normal individual during a period of 12 days on a normal diet consumed in the food 147.8 mg. of iron; in the urine and faeces 148.5 mg. of iron were found. This amount is not a measure of the absorption, for a good deal of the consumed iron was never converted into absorbable iron, but passed through the intestine and was found in the faeces. This balance between intake and output of iron is in agreement with the statement above, that the daily intake and excretion are normally about equal. After four days on such a diet, in another case, 50 mg. of iron were added daily for 10 days (a total of 500 mg. of iron), and the diet was then continued for five days. The total iron output was 24.2 mg. less than the total consumed iron. This quantity is a measure of the iron retained in the body. In the following Table is given the retention when one administration of iron was given on the fourth day during a ten-day feeding experiment on a normal diet. By such experiments, Lintzel determined in man the "iron retention" during the administration of iron preparations.

TABLE.

Compound.	Retention Experiments. Amount of iron added in mg.	Iron retained mg.
Ferric chloride ..	50	15.6
Do. do. .	50	14.7
Ferrous chloride with sodium chloride	50	13.6
Do. do. ..	50	15.1
Ferrous sulphate .	50	15.6
Ferrous lactate ..	50	16.3
Spinach cooked ..	28.1	11.5
Do. do. ..	18.4	6.8
Cabbage	17.5	7.0
Haemoglobin	130	1.7
Do.	50	0.1
Do.	50	1.7
Potassium ferrocyanide	50	1.3
Ferrous lactate with sodium lactate	50	0.4

From Lintzel's experiments with food, and from these retention experiments, it is evident that the normal individual may easily obtain adequate iron from various iron salts, or from some vegetables, such as cabbage or spinach (a pound of spinach has about 35 mg. of iron). Haemoglobin is a very poor source for iron, and so is the form of iron contained in muscle. It will be noted that complex salts which contain iron in the anion, and salts which ordinarily will dissociate but are prevented in this by the presence of an excess of anion, are also very poor sources of available iron.

Experiments on animals have shown that during a normal diet or during the administration of simple iron salts, the villi in the upper portion of the intestine contain iron in an ionized state, for it blackens when treated with ammonium sulphide. The normal individual on an ordinary diet is able in some manner to obtain his iron requirement from his food; but when, however, some pathological condition arises, causing anaemia, this amount is often insufficient to maintain the haemoglobin level, and iron preparations must be added to the diet. The most commonly used are ferric chloride as the tincture or solution, reduced iron (metallic iron), iron and ammonium citrate, ferrous sulphate (Blaud's pills), ferrous chloride, mineral spring waters. Frequent blood (as blood sausage, or taken as a drink, because of a popular belief in its use as a blood builder), and foods rich in iron (egg yolk, spinach, etc.) are given in abundance.

The literature contains clinical evidence which shows that reduced iron in doses of twenty to forty grains daily, is very efficient in producing haemoglobin. Bauer, by means of in vitro experiments with various acids acting on reduced iron, has recently demonstrated the reaction which renders metallic iron so valuable therapeutically. Hydrochloric acid in particular rapidly attacks it, forming ferrous chloride. The rate of conversion of the metallic iron into ferrous chloride is increased by increasing the exposed surface of the iron, i.e., by larger doses; also by increasing the concentration of the acid when acting on small amounts of iron. Organic acids likewise react with reduced iron, forming soluble salts, but not so energetically. This reaction depends on individual acids, the pH of the acid and the reacting surface of the iron. A fact which must be noted here is that no ferric salts can form so long as metallic iron is present in an acid medium, for it immediately reduces them to the ferrous condition. As ferrous salts do not react with proteins, they are not precipitated in the stomach, and pass into the intestine, where they are absorbed. McLendon has shown that the upper third of the small intestine is acid in reaction, and therefore in this area ferrous salts will be in an ionized state and ferrous iron ions readily pass into the gut epithelium. This has been proved histochemically by many observers (McLendon, Gaule, and others).

Sullman has pointed out that gastric and duodenal carbon dioxide play an important role in the production of soluble ferrous salts. Carbonates react readily with ferric salts to form insoluble ferric carbonate, which is not absorbable. Carbon dioxide will convert a small portion of this into the soluble ionized ferrous bicarbonate. Under most favourable conditions in vitro, as much as 64 mg. of ferrous iron per 100 cc. have been produced as bicarbonate.

Iron and ammonium citrate is a complex salt which does not give the ordinary inorganic tests for iron, because the iron is locked up in the complex anion. In vitro, a few drops of dilute hydrochloric acid rapidly splits off iron from this salt in ionized form, which gives a ferric ion test.

The tincture or the solution of the perchloride of iron contains iron in a soluble form in which practically all the ions are in the ferric state.

The iron in foods, such as egg yolk, spinach, cabbage, etc., is in the ferric state, and so is the iron in haemoglobin

when it is fed in the form of blood. During gastric digestion these various forms of organic iron compounds are partly split up, and some iron is liberated in the form of soluble ferric salts which give rise to ferric ions. The amount of available iron will vary according to the quantity and quality of the food. Eggs, spinach, etc., appear to be fairly good sources of iron, but haemoglobin is not, as was shown above. By means of an artificial gastric digestion of haemoglobin, Barkan and Lintzel have shown that in the time corresponding to normal digestion, 5% of the iron becomes ionized. With the ordinary diet, the amount is about 10% to 15% of the organic iron consumed. In ruminants, 50% of the iron consumed appears as soluble ferric iron.

Keeping in mind that all forms of organic iron in food give rise to ferric ions during digestion, and that the two iron preparations above, iron and ammonium citrate and ferric chloride, also have their available iron in the ferric state, it is well to observe what happens to ferric ions in the stomach and intestine. These ions react with proteins very rapidly, and in the course of digestion or when ferric salts are added, many of the ions must form insoluble proteinates which pass through into the intestine, where, in the alkaline portion, they are precipitated as a hydroxide or sulphide. None of these, therefore, will be available for absorption. Sullman has shown by his experiments, however, that there are numerous reducing substances in the chyme and digestive juices which reduce ferric ions that have not united with protein to the ferrous state. Even moistened bread will reduce ferric chloride to ferrous chloride. Sulphydryl bodies, the amino-acids tyrosine and histidine and glutathione which is present in the stomach mucosa, will, in some measure, furnish some ferrous ions from ferric salts which are still in solution in the acid content of the stomach and upper tract of the intestine. But one must realize that the amount of ferrous iron available for absorption will be but a small percentage of the ferric iron split off from food iron or complex salts administered, or even from large amounts of ferric chloride solution given after meals.

Ferrous salts have distinct advantages over ferric, in that they are not so irritant to the stomach mucosa and do not give rise to so much gastric disturbance. They do not react with proteins, and in the acid content of stomach and intestine remain in a soluble state in a completely ionized form. The available ions do not depend upon gastric acidity (hydrochloric acid or organic acids) when ferrous salts are given per os. Ferrous chloride appears to be more efficient than ferrous sulphate. It is more soluble in lipoids which may account for this difference. Mineral spring waters contain iron salts usually as ferrous bicarbonate. They have a wide reputation in the treatment of anaemia, but as the quantity of iron is small, much water must be consumed.

The absorption of iron may take place from any portion of the gastro-intestinal tract, but owing to the fact that the ionized ferrous salts are the only ones available for absorption, and these can exist only in the stomach and upper third of the small intestine, normally the absorption is confined almost exclusively to this area. Lintzel has shown when the iron intake is low the epithelial cells in the gut are poor in iron and greedily take up traces of available iron. When the intake is high, the cells first take up to their capacity, then become plugged, as it were, and do not take up as much iron, even although it is available. In a series of experiments on man, Lintzel found that the retention was as great after feeding 50 mg. of ferrous sulphate as it was after 200 mg.

Iron is excreted mainly through the small intestine. A small amount of iron (1 mg. or less) appears in the urine and the large intestine excretes about one-seventh of the total excretion. When simple ferrous salts are injected intravenously, about 3% appears in the urine and the remainder is excreted

by the gut. Complex salts, such as sodium iron citrate, intravenously, differ, however, in that as much as 75% may be excreted in the urine. When ferrous salts are given per rectum in rabbits, if the stomach and intestines are observed at the same time, it will be noted that iron is being excreted. Such is the rapidity with which iron may escape from the body.

The storehouses for iron in the body are the liver and the spleen. The spleen per gram has a greater amount of iron, but owing to its size, the liver contains the greatest weight of iron. The transport of iron from the gut epithelium is not understood, and it is of no avail to report theories. Starkenstein has shown that absorbed ferrous salts are changed to ferric in blood serum, and as such must be carried to the liver and spleen. Because of the small amount of iron in the serum at any given moment, it is difficult to determine the amount quantitatively. How the stored iron is later made available for haemoglobin formation is a question as yet unanswered. This problem of transport of iron and the building into haemoglobin is still awaiting some wide-awake physiologist or pharmacologist for solution.

The total amount of iron in the blood of man is quite small. Minot and Murphy have stated that per 100 cc. of blood in males, there are 44.8 mg. of iron; and in females 42.5 mg. Assuming a woman to weigh 55 kg. (120 lbs.), and that one-thirteenth of the weight is blood, the 4.2 kg. of blood would contain 1,800 mg. of iron, i.e., about 275 grains. A 70 kg. male would have about 2,500 mg., or a little less than 400 grains.

Judging from the clinical reports of Hare, Hurst and Dewitts, massive doses of iron preparations greatly increase haemoglobin production in women suffering from late chlorosis or chronic idiopathic hypochromic anaemia. If one may assume that these women would have 1,800 mg. of iron with a normal haemoglobin, a simple mathematical treatment of the figures presented by these clinicians show some interesting facts. One patient reported by Hare, with a haemoglobin of 44% after 19 days on 90 grains of iron and ammonium citrate daily, showed a rise of 58%. This means that 254 mg. of iron were transformed into haemoglobin in the 19 days, or that per diem 13 mg. were absorbed and converted to haemoglobin. In the next four months the haemoglobin rose to 96%, which means a daily conversion of 6 mg. For the next three months the daily conversion was 1.5 mg. A large number of cases show similar figures. The initial conversion varies from 10 to 50 mg. daily; as the haemoglobin increases, the daily conversion gradually drops to 1 or 2 mg., even though the dose of iron salt is still as high as it was in the beginning.

It is evident that in the early stages of iron medication in these anaemic patients that the intestinal epithelium is rapidly absorbing iron and forwarding it to the liver and spleen. As the haemoglobin increases, the rate of absorption slows up, or the iron is not so rapidly transported from the epithelium, which becomes plugged with iron. The demand of the body for more iron to form haemoglobin may control the rate of iron absorption, and although the same large dose may be given, smaller and smaller quantities find their way into the blood.

In a recent paper, Reimann and Fritsch have reported increase in haemoglobin and reticulocyte stimulation in secondary anaemia following small doses of ferrous chloride, which are comparable to that obtained by massive doses of other iron salts. The amount of ferrous chloride they gave per diem is about 2 1-4 grains, which is equivalent to 100 mg. of iron, and appears to have as much effect as 20 to 40 grains of reduced iron or 90 to 120 grains of iron and amonium citrate. Medication with this salt relieves the patient of any irritant gastric disturbance, and the constipation that accompanies massive iron treatment.

The necessity of adding small

amounts of copper to massive doses of iron is questionable. In the study of nutritional anaemia of rats, Waddell, Steenbock, Hart, Whipple and others have shown clearly that iron salts freed from copper with hydrogen sulphide are useless in preventing or curing this anaemia. It is difficult to choose a copper-free diet—any man may find enough copper for his wants in the normal diet, whereas certain lower animals may not. However, Mills of the Montreal General Hospital reported that certain cases of hypochromic anaemia, which would not yield to massive iron treatment, made excellent progress when 0.05 g. of copper sulphate was added to the iron. The daily conversion of iron to haemoglobin was about 18 mg. Mills' work, unfortunately, is open to criticism, because when his patients failed to respond to 60 grains of Blaud's pills daily, he changed to 90 grains and added the copper. His copper effect may be due to the additional iron salt. Adamson and Smith in a later paper reported the necessity of copper along with 90 grains of Blaud's pills in secondary anaemia.

When given subcutaneously, two types of iron only show remarkable toxicity, namely, the ferrous salts and several complex salts, such as sodium iron citrate. This latter salt, according to Starkenstein, is not an organic form of iron, although iron is held in the complex anion. Organically bound iron is united directly to a carbon or a nitrogen. The toxic effect of the two types are quite different. The ferrous salts in rabbits and dogs in toxic doses produce a narcosis resembling that produced by magnesium sulphate. The animals lie on their sides and reflexes are still present. Later they get up spontaneously, and then after an hour or so the second toxic symptoms rapidly appear, which consist of convulsions of a tonic-clonic type, and finally death. The ferrous salt effect is counteracted by calcium chloride. In dogs, large doses per os or small doses subcutaneously or intravenously give rise to vomiting. Vomiting is a characteristic of poisoning by ferrous sats. It may be of interest to note that in a case reported by Hurst, of apparent iron poisoning in a woman (200 grains of iron and ammonium citrate per os in one day), vomiting continued at intervals for several hours.

The complex salts in small doses in rabbits and dogs produce a paralysis of the motor nerve endings, which causes death in a day or so. Larger doses are fatal in an hour. After intravenous injection, a marked diuresis occurs in rabbits, and following this or a subcutaneous injection, diarrhoea sets in. After toxic doses, dogs become feeble, lie in their cages, reacting but little to stimuli. They will not bark or eat. If food is given, it is vomited. The urine may contain a trace of protein. After death, the liver and kidneys show marked fatty changes. If the dogs do not die from the dose, they recover completely in 4 to 6 days.

In frogs, ferrous salts and complex salts show much the same effect. The toxicity of both these types of salts varies with the anion, the malate being less toxic than the tartrate and citrate, but much more than the chloride. Ferrous salts on the isolated heart and intestines show a depressant effect which is removed by caffeine. The complex salts on the intestine have a slight stimplant effect. Ferrous salts cause a marked, but temporary decrease in blood pressure. Complex ferrous and ferric salts show the same action.

Soluble ferric salts in the ionized state, when injected subcutaneously, show no effect other than gross local damage. Intravenously, they cause death at once, because of their protein precipitant effect. In the stomach they are irritant and give rise to gastric disturbances.

The toxic action of the above-mentioned types of iron salts is not understood. The ferrous salts, it is believed, are rapidly oxidized in the blood stream to ferric, and then combine with the proteins of the serum, after which they are held by the liver. Ferrous salts per

rectum in rabbits and rats are carried into the iliac veins and do not reach the liver as rapidly as when absorbed from the small gut. They show much greater toxicity when given thus. The complex salts do not react with proteins, and are free to circulate as such. Pharmacologists have still to unravel the problem of their toxic effects.

In conclusion, two outstanding characters of iron salts may be cited. (1) Ferrous chloride in small doses appears to be as efficient in the treatment of some anaemias as massive doses of other iron salts. (2) Pharmacologically, two types of iron salts are active—ferrous salts and inorganic complex salts.

Electric Shock.

By ARNOLD FARRELL, '32.

Since 1879 many researches have been undertaken to discover, if possible, the cause of death in electric shock, and to formulate suitable remedial measures to protect those exposed to the possibility of such accidents.

Jex Blake, in 1913, reviewed and summarized the whole field of electric shock up to that time. He reached the following conclusions:

1. The existence of an idiosyncrasy to electric currents in man is doubtful.
2. Death in human beings may occur in different ways:
   (a) by a prolonged tetanus causing death by asphyxiation;
   (b) by primary heart failure or ventricular fibrillation;
   (c) by failure of respiration, either through nerve inhibition or through actual damage to the central nervous system;
   (d) by delayed death, the result of burns or complications.
3. The electrical details are of great importance. With voltages over 500 the brain and spinal cord are more often affected, while with lower voltages the heart is the usual site of action.
4. Artificial respiration is recommended as the only method of treatment holding out any hope.

Jellinek(2) stresses the importance of immediate and continued application of efficient artificial respiration, particularly when the "apparent death" is due to respiratory failure. He believes that if fibrillation of the heart is present the slight massage afforded by this measure may have some beneficial effect on the patient.

Scott Ram(6) made an analysis of the records of electric accidents in England from 1912 to 1921. He points out that of those who had received over 650 volts, 62.5 per cent recovered, while only 39 per cent. of those who had received under 250 volts were saved.

The records of electric accidents in Ontario to some extent confirm these observations.

Urquhart(7), working in Laboratory of Physiology, University of Toronto, using rabbits because of the relative difficulty of throwing a rabbit's heart into fibrillation, found that the results varied considerably according to the parts of the body to which the electrodes were applied, and according to the exact design of the electrodes. In his first series of experiments he reached the following conclusions:

1. The cause of death in electric shock may be primary cardiac failure, or primary respiratory failure, or a combination of both.

2. In laboratory animals, when the current traverses the body from the

head to a hind limb, about 45 per cent. of the deaths are of purely cardiac origin. The remainder of the deaths occur because of failure of the respiratory movements. It is to be noted that the ratio of 45|55 per cent. may be somewhat related to that of Scott Ram's figures which have been quoted above.(6)

3. In these, and in the group in which the current is passed directly through the brain, a condition of profound paralysis or block becomes established in the respiratory, vagus and vasomotor centres.

4. That the block involves these centres is shown by experiments, which demonstrate clearly the absence in electrocuted animals of reflex effects normally functioning through them. Since the nerve centres become insensitive to extraneous influences, "counter-stimulation" (i.e., stimulation of sensory nerves by pressure or blows) is not an aid to recovery.

5. No definite histologic changes can be made out in the brain to account for the symptoms. The capillary hemorrhages which occur do not appear to be significant.

6. When the electric current does not cause charring of nerve structures the paralysis or block is recovered from and the reflexes return, provided efficient artificial respiration is applied.

7. It is of the greatest importance that artificial respiration be applied early and be maintained for a sufficient length of time. Owing to the nature of the block in the nerve centres the ordinary tests for death should not be accepted, and nothing less than cooling of the body or the onset of rigor mortis should be considered to be evidence of death.

Urquhart(8), in a second series of experiments, demonstrated that a temporary block could be produced in the spinal cord or in a nerve trunk by the passage of an electric current.

Kouwenhoven and Langworthy(9), applying electrodes to the head and tail of rats, found:

1. A 1,000-volt continuous current was more dangerous to rats than an alternating current of the same effective voltage.

2. A large rat survived a greater shock than a small rat.

3. Both sexes were equally susceptible to electric shock.

4. Paralysis of the hind legs resulting from hemorrhages in the spinal cord characterized many of the A-C experiments.

Their more recent experiments confirm Urquhart's(7) conclusion that when an electric current passes through the brain a temporary physiological block is produced in the respiratory centre, and that spontaneous breathing ceases for a time; also, that if no serious injury to the heart occurs, adequate artificial respiration may give time for the centre in the medulla to recover and for normal breathing to begin.

Maclachlan(11) reports a case of recovery from shock following eight hours' of continuous application of the Schaffer prone pressure method, which also confirms Urquhart's findings.

It was recognized that the D.C. shocks more often produced symptoms of irritation to the nervous system, such as convulsions, whereas many of the rats receiving A-C shocks showed paralysis of the posterior portion of the body directly after the shock. These paralyzed animals were quiet, would not eat, and never lived longer than two or three days.

Histo-Pathology of Nervous System Following Electric Shock.

These latter cases were studied after death showed an abnormally soft spinal cord; and microscopic observation of cross-sections of the cords revealed that an area of hemorrhage, extending over a relatively long portion of the cord, had severed nervous pathways. These hemorrhages were in the central region of cord. Similar hemorrhages, but smaller, occurred in the region of the brain. When these existed in the region controlling respiration, they were considered responsible for the death of the animal. In some cases

hemorrhages were present in the meninges also.

In the cases of experimental animals, hemorrhages in the nervous system are responsible for the immediate or subsequent death of a certain group subjected to contacts with electric circuits. They are much more common after contact with A-C circuits than after contact with D-C circuits; and they increase in number with the increase of circuit potential.

Hemorrhages have been found also in the brains of men who have died as the result of similar shocks; such hemorrhages probably are caused by the sharp rise of blood pressure which follows the breaking of contact with an electric circuit.

During the period of current flow, the blood collects in the veins, being squeezed from the heart and arteries by the contraction of the muscles in their walls. When the heart resumes its beating all this blood is forced back at once into the heart and arteries, often over-taxing the capacity of the arteries and resulting in rupture. With either A-C or D-C 1,000-volt circuits, severe burns often occurred around the electrodes, in some cases resulting in an erosion of the skull beneath the head electrode, and even burning the surface of the brain itself. Generally burns from contact with a 1,000-volt D-C circuit were found to be more severe than those from a 1,000-volt A-C circuit.

It was found that all nerve cells, even in a given group, did not show an equal amount of damage, normal cells often being found among others that had suffered great changes, the nuclear edges being indistinct, the nucleolus swollen, with a loss of granules from the centre of the cell. Extensive microscopic studies support the belief that cells having the potentialities of recovery may be differentiated from those rendered completely and permanently functionless.

In this connection the nucleus is the real index of the ultimate fate of the cell; if it shows marked damage the cell will die.

It is important to note that only those nerve cells in that portion of the nervous system traversed by the current were damaged.

The type of change produced in nerve cells by electric shocks is non specific, and may be caused by other physical and toxic agents. Very high body temperature may damage nerve cells, and it is an established fact that body temperature rises rapidly in the case of electric shock if the circuit is closed for any length of time. This is supported by the results of autopsy performed on a man who suffered accidental death by contact with a 2.2 kv. 60-cycle circuit. The contact was from one arm to one leg, lasted several minutes, and badly burned the body. In this case the brain cells showed marked changes, even though that region did not lie in the direct pathway of the current. Therefore it must be considered that if the body temperature is elevated to a considerable degree, changes everywhere in the delicate nerve cells must be expected. These changes are indistinguishable from those produced by electricity alone.

In addition to heat, there is another cause which affects nerve cells lying outside the current path. Where, for example, the contacts are at points on the same arm or leg, the passage of the electric current between these points gives rise to violent impulses which the peripheral nerves transmit to the brain. When these impulses are very strong they may produce a condition of shock akin to that perceptible after operations and fractures.

Apparatus was devised to eliminate heat as a source of nerve cell damage. Marked changes in the nerve cells themselves were found in the brain and spinal cord only when they were traversed directly by the surge current. It is clear, therefore, that an electric discharge produces profound changes in nerve cells, even in cases where heating is not a possible factor. When the period of contact is prolonged, heat undoubtedly produces marked alterations.(13)

Discussion and Conclusions.

1. A temporary respiratory block is produced, the location of which depends upon the current path (and strength of voltage) in the brain stem or the vagus nerves. Resuscitation is possible provided the damage done does not require a recovery time too long to permit life to be supported in the interval.

2. In laboratory animals, when the current traverses the body from the head to a hind limb, about 45 per cent. of the deaths are of purely cardiac origin. The remainder of the deaths occur because of failure of the respiratory movements.

3. A 1,000-volt continuous current circuit is more dangerous than an alternating circuit of the same effective voltage, irrespective of the current path.

4. The chances of recovery are best when the brain does not lie directly in the pathway of the current.

5. The presence of a respiratory block has been demonstrated, and the fact that when natural breathing is first established the animal is not necessarily on the road to complete recovery.

6. The respiratory block is less severe, if not absent, when the current traverses the posterior portion of the body.

7. Burning of the tissues with continuous current is much more severe than occurs for the same effective alternating current.

8. If the body temperature is elevated to a considerable degree, changes take place in nerve cells indistinguishable from those produced by electricity alone; these occurring in severe electric burns, and the body temperature rises rapidly in the case of electric shock if the circuit is closed for any length of time.

9. Hemorrhage may take place into the area of brain controlling respiration and cause death.

10. Artificial respiration should be applied early, and kept up until body cools or rigor mortis develops.

Treatment.

1. Clear patient and start resuscitation as soon as possible, according to the Schaffer's Prone Pressure Method.

2. Keep the patient warm.

3. Since the paralysis of the breathing may last for some time, continue artificial respiration until it is absolutely certain that the heart has ceased beating, or rigor mortis develops. Oxygen, carbon dioxide, heart stimulants, or pulmotor are of no advantage as judged by experiments on animals.

4. After natural breathing returns the patient must be kept lying down, and he must be carefully watched for several hours to see that the paralysis of breathing does not return. If it does, artificial respiration must be reapplied.

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Modern Conceptions of Epilepsy.

G. E. HOBBS, B.A., '33.

Epilepsy is defined as a syndrome characterized by a sudden appearance of paroxysms of which convulsive movements, or loss of consciousness, or both, are the principal element.

The paroxysmal attacks are one of two types, the "grand mal" and the "petit mal."

The "grand mal" is the classic major epileptic attack, and consists of four definite stages. There is a cry with sudden and complete loss of consciousness, with the patient falling to the ground without making any movement to protect himself, so that injury is common. Following this there is immediately a tonic spasm of all the muscles in the body. During this cyanosis may develop as respiration ceases, due to spasm of the muscles of respiration and a closure of the glottis. This stage of tonus is then replaced by a clonic jerking of the muscles. The movement of the muscles to the jaw may cause the tongue and cheeks to be bitten. Urine and faeces may be discharged involuntarily. Following this there is a stage of coma, where the patient remains in a condition like sleep for a varying length of time. After recovering from the fit, the patient is somewhat bewildered, and complains of being tired, but his mental condition is usually better than before the fit.

The stage of tonus lasts about 1 min., the clonic stage 4 mins., and the coma an hour or two.

During the attack one commonly finds a positive Babinski, loss of corneal reflex, and first contraction then dilatation of the pupil.

The "petit mal" consists of transient unconsciousness, which may come on with or without a feeling of faintness and vertigo.

An epileptic attack may or may not be preceded by a localized sensation known as an "aura." The most common of these are a pain, sensation of warmth or cold, or a sinking sensation in the epigastrium.

The frequency of the occurrence of seizures caries greatly. Occasionally they are so frequent as to succeed one another without intermission. This condition is known as "Status epilepticus," and may go on until exhaustion and death.

Patry, in a study of 31 cases, found a marked difference in the diurnal and nocturnal incidence of their convulsions. Approximately 2/3 of these concentrate their attacks at certain hours. Many show a marked periodicity of attacks.

Between the attacks the person affected usually shows transitory disturbances of mood, being excessively irritable, and picking a quarrel with great facility. These phenomena have been called the "epileptic character."

Robb has shown also that the reaction time is poor, and that the epileptics are less precise in their movements than normal persons.

Etiology.

When an attempt is made to determine the factors behind the paroxysmal seizures, we find they are divided into two large groups: (1) Organic; (2) Essential epilepsy.

n organic epilepsy a large variety of pathological lesions are seen, such

as brain tumour, arteriosclerosis, traumatic lesions, etc., which may account for the seizures. However, it appears that these lesions do not represent the whole story, for Parker, in a survey of 313 cases of intracranial tumours, all of which were above the tentorium, found that only 21.6% developed major epileptic seizures. Also, out of 18,000 cases of gunshot wound of the head, less than 5% developed epilepsy. So that undoubtedly these pathological lesions do play a part, but the greater part played by some inherent capacity of the patient to respond to that type of stimulation with a convulsion.

In essential or idiopathic epilepsy no gross anatomical change in the main can be shown, so that effort must be made to account for this along different lines.

A considerable mass of evidence has accumulated to show that when convulsions occur the body is in a state of alkalosis. Lennox reports a number of cases showing a close relationship between occurrance of the symptoms and plasma bicarbonate. The following case is typical of his findings. This is a girl age 17, who has had from 5-20 petit mal attacks daily for years. In the experiment the number of attacks was recorded, while attempts were made to change the reaction of the body in various ways. This change in reaction of body was estimated by measuring the level of plasma bicarbonate. Ammonium chloride was taken and the plasma bicarbonate fell from about 65 vols. % of CO_2 to about 50. There was a corresponding fall in the frequency of convulsions from about 15 per day to 3 or 4. Alkali was then taken for two days, and the plasma bicarbonate rose to about 85 vols. %, and the convulsions increased accordingly to 27 per day. Following this the patient fasted for 16 days. The plasma bicarbonate fell to about 45 vols. %, and there was complete disappearance of convulsions. The patient remained free of attacks for a week after resumption of a carbohydrate diet in spite of the fact that the concentration of plasma bicarbonate had returned to normal. Several hours after injestion of 25 gms. of alkali, however, seizures recurred with greater frequency than she has ever had them before. When the patient was given a fat diet, and plasma bicarbonate had again fallen, seizures again disappeared. Lennox failed to find any relation between the water balance and the frequency of seizures. Thus from this work it would seem that the frequency of epileptic seizures varies in proportion to the alkali reserve. To support this view, there are numerous reports of beneficial results of the production of an acidosis by feeding a diet high in fats and thus producing an acidosis. The increase of nerve irritability by the alkalosis may be a factor.

Much evidence has also accumulated to show that there is a disturbance in the water metabolism during spilepsy. Gamble studied the water output of patients with epilepsy by studying the excretion of fixed base, since the water excreted is accompanied by a parallel withdrawal of fixed base. He found that during the seizure there was a great increase in the fixed base excretion with a decrease after the seizure. This was explained as being due to an alternate release and retention of body fluid. Since the fixed base was mainly sodium, he concluded that there was a tendency for the extracellular water to be excreted. McQuarrie showed fairly conclusively that there is a close relationship between the water balance and the occurrence of symptoms. He cited numerous cases where, if the water intake was limited, there were no seizures, but as soon as the intake rose above a certain value, convulsions occurred. Also, if diureses follows the seizures it favours the prevention of further seizures temporarily. He believes the fundamental factor in the production of seizures is a disturbance in the water balance affecting the central nervous system more specifically. In support of this, T. Fay reports very favorable effect of fluid limitation in

epileptics. His experiments cover a two and a half - year period.

Also, Rowntree has shown in experiments on animals that an excess of water brings about violent convulsions.

The supporters of the idea that the convulsions are as a result of a disturbance in water balance, explain the increased occurrence of seizures with administration of alkali as due to the fact that an increased alkali reserve favours the accumulation of fluid within the body. If we accept this theory it also explains the favourable results reported following venous section, fevers, and a large variety of surgical operations performed on epileptics.

The acid base balance and water balance doubtless play a part in the seizures of idiopathic epilepsy, but these changes, in themselves, are not great enough to bring about this phenomenon. Thus to complete the picture we must, as in the case of organic epilepsy, postulate some inherent tendency towards convulsive seizures.

The question now comes forward: Is this inherent tendency hereditary? At our present state of knowledge on the subject we can not definitely say. Burr, after a study of 1,449 cases, concludes that there is no evidence to support the supposition that epilepsy is directly hereditary. The frequency of insanity, crime, chorea, alcoholism and epilepsy in relatives of epileptics point toward congenital instability resulting from abnormality in the germ cell. His conclusions are, in other words, that a predisposition to nervous and mental disease is inherited, but the resulting specific disease depends on the external causes.

Some investigators hold the view that the explanation of the origin of the convulsive seizures is not to be found in the physical realm, but in the psychical. As already pointed out, certain characteristic mental reactions are found in epileptics in the period between the seizures which constitute the "epileptic character." For a considerable length of time this epileptic character was looked upon as the result of the effect of these seizures upon the personality. It is now held by many that this type of personality is frequently demonstrable in the individual before seizures occur. The occurrence of seizures is then explained as an inability of the patient to adjust to the environment. Clark describes the epileptic as an "egocentric, emotionally inexpressive individual involved with excessive worship of self which produces an inelasticity of the personality incompatible with flexible living, hence the explosive fits."

Treatment.

The first thing to be done in treatment is to put the patient in as good physical condition as possible by removal of any foci of infection, and with the proper proportion of work or exercise to rest.

Also, an effort should be expended so that the patient should not be allowed to center his attentions upon the convulsive seizures, but whenever possible should see themselves as useful members of society. They should be protected from emotional strain and excitement.

In a few patients cessation of seizures follows the above procedures.

As a means of treating the symptom phenobarbitol has gradually replaced bromides, due to the fact that it does not so greatly depress the mentality. The time of giving the dose should vary with the regular appearance of the symptoms. Thus if the seizures occur most frequently during the night the drug should be taken after the evening meal; with patients having occasional spells at difinite times, 1½ grains a day is usually enough, but if the symptoms are more frequent and severe, then 2 - 2½ grains may be given. If this is not enough, the drug may be given three times a day, starting with ¾ of a grain and increasing, if necessary, to even 3 gr. three times a day.

Once the proper dose is established then this should be continued indefinitely. In a few cases it may be with-

drawn slowly, particularly with children after puberty and young adulthood is passed. Petit mal attacks are difficult to treat with phenobarbitol.

The form of treatment that has aroused the greatest interest in the past few years is the ketogenic diet originally introduced by Peterman. In giving the diet, he believes that the total calories given should not exceed the total requirements. Usually 15 gm. of carbohydrate are given with one gram of protein per kilogram of body weight and enough fat to make up the caloric requirements. This diet produces a state of profound acidosis. The most favourable results were obtained in cases of petit mal which do not respond well to phenobarbitol. He reported that 10 out of 17 were free of symptoms. Peterman likens the action of aceto-acetic acid to an anaesthetic which will relieve convulsions for some time.

Since the original introduction the ketogenic diet has been tried by a large number of workers over a lengthy period of time and the majority of the results are favourable, reporting cessation of seizures in up to 30-50% of the cases treated. This diet was believed to correct an undue presence of alkalosis and to owe its beneficial effects to this fact.

As already pointed out, there appears to be a close relation between the water balance and occurrence of symptoms. Temple Fay reported treatments by fluid limitation extending over a period of 2½ years. He found that attacks of a grand mal nature were influenced or controlled by a restricted fluid intake where there was true cooperation from the patient. Also he found that attacks occurred when there were fluid indiscretions. McKuarrie also has demonstrated the relation of water balance to convulsions. He concludes that the beneficial results from fasting and ketogenic diets lead to an acidosis which favours a loss of water from the body.

Since the vital factor in the prevention of seizures appears to be to obtain a dehydration of the body tissues, it is now thought that this can best be obtained with a less rigid ketogenic diet accompanied by a limitation of the fluid intake.

All the work discussed so far in the treatment has been carried out on idiopathic epilepsy. However, Palmer claims to have obtained beneficial results from the treatment of traumatic epilepsy with ketogenic diet and fluid limitations.

In closing it is of interest with the new regime of ketogenic diets and dehydration to note that Hippocrates, or one of his school, on a treatise on epilepsy, made the following statement: "But whosoever is acquainted with such a change in men, and can render a man lumid or dry, hot or cold, by regimen, could also cure this disease."

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Why has not halibut liver oil been available to the medical profession before?

ANSWER:

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QUESTION:

What dosage is recommended?

ANSWER:

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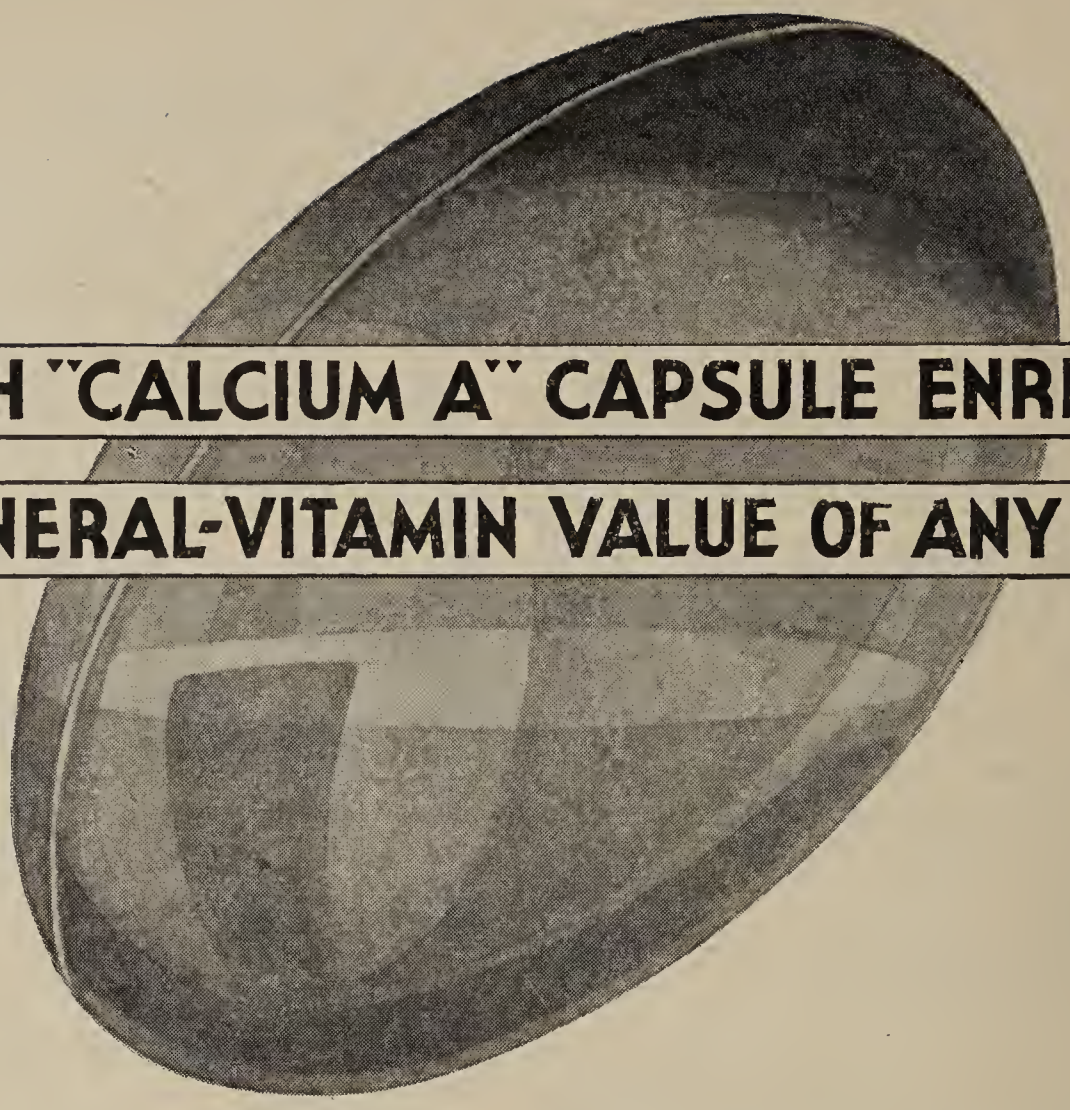

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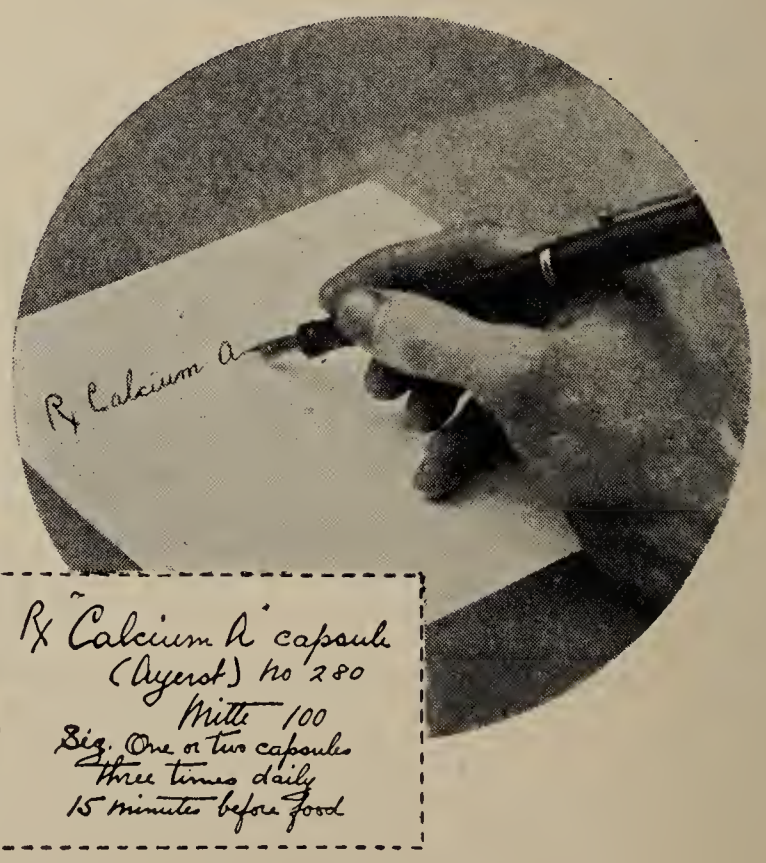

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